There are two genetic mechanisms that give rise to the Rhnull phenotype: regulator and amorphic. In the regulator–type Rhnull a mutation occurs in the RHAG gene, resulting in no expression of the RhAG protein and no RhD or RhCE protein expression on the red cells, despite normal inheritance of RHD and RHCE genes. These individuals can pass normal Rh genes to their offspring. In the amorphic–type Rhnull, there is a mutation in the RHCE gene inherited from each parent and the common deletion of the RHD gene found in most individuals. These individuals have inherited a normal RHAG gene. Rhnull individuals of either type lack the high prevalence antigen LW and FY5, an antigen in the Duffy blood group system. Antigens found on glycophorin B (S, s, and U) may also be depressed. Individuals of the Rhmod phenotype have partial suppression of the RH gene expression caused by mutations in the RHAG gene. The red cells of these individuals possess other blood group antigens, but may show weakened expression of Rh and LW antigens and depressed expression of S, s, and U antigens. These individuals may exhibit features similar to Rhnull syndrome, but their symptoms are less severe and .rarely problematic clinically